CHAPTER 40

The First and Second Heart Sounds

KEY TEACHING POINTS

- The most important characteristic of S_1 is its *intensity.* A *loud* S_1 indicates a vigorous ventricular contraction, short PR interval, or both. A *soft* S_1 indicates a feeble ventricular contraction, long PR, or both.
- If the pulse is regular and S_1 intensity *varies* from beat to beat, the only possible diagnosis is atrioventricular dissociation (e.g., complete heart block).
- The most *important* characteristic of S_2 is *splitting,* which may be normal (single or physiologic) or abnormal (wide physiologic, fixed, or paradoxic). The most common causes of wide physiologic or paradoxic splitting are the bundle branch blocks.

INTRODUCTION

The first and second heart sounds (S_1 and S_2) define systole and diastole and therefore form the framework for analyzing all other auscultatory physical signs, including the third and fourth heart sounds, clicks and ejection sound, knocks and opening snaps, and systolic and diastolic murmurs. In his classic treatise describing the discovery of the circulatory system, written in 1628, Harvey described both S_1 and S_2, comparing them to the gulping sound made by a horse drinking water.[1] The first person to state that S_1 and S_2 were the sounds of closing heart valves was Rouanet of France, who wrote in his 1832 MD thesis that S_1 occurred when the atrioventricular (i.e., mitral and tricuspid) valves closed, and S_2 occurred when the semilunar (i.e., aortic and pulmonic) valves closed.[2]

THE FIRST HEART SOUND (S_1)

I. THE FINDING

S_1 is heard well across the entire precordium, both with the bell and diaphragm of the stethoscope. It is usually loudest at or near the apex and contains more low-frequency energy than does S_2, which explains why, when mimicking the sound, the term *lub* is used for S_1 and the sharper term *dup* for S_2.*

* It was Williams in 1840 who invented the *lub dup* onomatopoeia.[3]

II. PATHOGENESIS

A. CAUSE OF S_1

The precise cause of S_1 has been debated for decades. Although its two recordable components coincide with closure of the mitral and tricuspid valves, the force of valve closure itself is insufficient to generate sound.[4] Instead, their closure probably causes moving columns of blood to abruptly decelerate, which sets up vibrations in the chordae tendineae, ventricles, and blood as a unit (i.e., **cardiohemic system**).[5]

B. INTENSITY OF S_1

The most important abnormalities of S_1 relate to its intensity; the sound can be abnormally loud, abnormally faint, or vary in intensity abnormally from beat to beat. The primary variables governing intensity of S_1 are strength of ventricular contraction and the position of the atrioventricular leaflets at the onset of ventricular systole.

1. VENTRICULAR CONTRACTILITY

The stronger the ventricular contraction, the louder the S_1. Strong contractions, which have a high dP/dT (i.e., large increase in pressure with respect to time), intensify S_1 because the valves close with more force and generate more vibrations in the cardiohemic system.[6-8]

2. POSITION OF THE VALVE LEAFLETS AT ONSET OF VENTRICULAR SYSTOLE

If the mitral valve is wide open at the onset of ventricular systole, it will take longer to close completely than if it had been barely open. Even this small delay in closure intensifies S_1 because closure occurs on a later and steeper portion of the left ventricular (LV) pressure curve (i.e., dP/dT is greater).[9]

The PR interval is the main variable determining the position of the valves at the beginning of ventricular systole. If the PR interval is short, ventricular systole immediately follows atrial systole (i.e., the R wave immediately follows the P wave). Because atrial systole kicks the valve open, a short PR guarantees that the valve will be wide open at the onset of ventricular systole. In contrast, a long PR interval allows time for the cusps of the atrioventricular valves to float back together before ventricular systole occurs. Studies show that, with PR intervals less than 0.20 seconds, the intensity of S_1 varies inversely with the PR interval (the shorter the PR interval the louder the sound). With PR intervals greater than 0.20 seconds, S_1 is faint or absent.[8-10]

III. CLINICAL SIGNIFICANCE

A. LOUD S_1

S_1 may be abnormally loud because of unusually vigorous ventricular contractions or because of delayed closure of the mitral valve.

1. VIGOROUS VENTRICULAR CONTRACTIONS

Vigorous contractions, such as those occurring from fever and sympathetic stimulation (e.g., beta-adrenergic inhalers, thyrotoxicosis), increase dP/dT and intensify S_1.[6]

2. DELAYED CLOSURE OF THE MITRAL VALVE

A. PROLAPSED MITRAL VALVE

In patients with the murmur of mitral regurgitation, a loud S_1 is a clue to the diagnosis of early prolapse of the mitral valve (many patients with mitral regurgitation have a normal or soft S_1).[11,12] S_1 is loud in these patients because the prolapsing leaflets stop moving and tense later than normal, when dP/dT in the ventricle is greater.[11]

B. MITRAL STENOSIS

Ninety percent of patients with pure uncomplicated mitral stenosis have a loud S_1.[13] Because the murmur of mitral stenosis is often difficult to hear, a traditional teaching is that clinicians should suspect mitral stenosis in any patient with a loud, unexplained S_1 and listen carefully for the murmur with the patient lying on the left side.

Mitral stenosis delays closure of the mitral valve because the pressure gradient between the left atrium and left ventricle keeps the leaflets open until the moment of ventricular systole. After successful valvuloplasty, the loud S_1 becomes softer.[13]

C. LEFT ATRIAL MYXOMA

Many patients with left atrial myxoma (seven of nine in one series) also have a loud S_1 because the tumor falling into the mitral orifice during diastole delays closure of the valve.[14]

B. FAINT OR ABSENT S_1

S_1 is unusually faint if ventricular contractions are weak or if the mitral valve is already closed when ventricular systole occurs.

1. WEAK VENTRICULAR CONTRACTIONS (LOW DP/DT)

Common examples of weak contractions causing a faint S_1 are myocardial infarction and left bundle branch block.[15]

2. EARLY CLOSURE OF THE MITRAL VALVE

Common causes of early mitral closure causing the faint S_1 include the following:

A. LONG PR INTERVAL (>0.20 SECONDS)

See the section on Intensity of S_1.

B. ACUTE AORTIC REGURGITATION

In patients with the murmur of aortic regurgitation, the faint or absent S_1 is an important clue that the regurgitation is acute (e.g., endocarditis) and not chronic. Patients with acute aortic regurgitation have much higher LV end-diastolic pressures than those with chronic regurgitation, because the acutely failing valve has not allowed time for the ventricle to enlarge, as it does to compensate for chronic regurgitation. The high pressures in the ventricle eventually exceed diastolic left atrial pressures, closing the mitral valve before ventricular systole and thus making S_1 faint or absent.[16]

C. VARYING INTENSITY OF S_1

If the arterial pulse rhythm is *regular* but S_1 varies in intensity, the only possible explanation is that the PR interval is changing from beat to beat, which means the patient has atrioventricular dissociation. In contrast, in patients with *irregular* rhythms, changing intensity of S_1 has no diagnostic significance, because ventricular filling and dP/dT—and therefore S_1 intensity—depend completely on cycle length.

In patients with pacer-induced regular rhythms, an S_1 that varies in intensity is compelling evidence for atrioventricular dissociation (LR = 24.4; see EBM Box 40.1).

EBM BOX 40.1
*The First and Second Heart Sounds**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding is	
			Present	*Absent*
First Heart Sound				
Varying Intensity S_1				
Detecting atrioventricular dissociation[17]	58	98	**24.4**	0.4
Second Heart Sound				
Fixed Wide Splitting				
Detecting atrial septal defect[18]	92	65	2.6	**0.1**
Paradoxic Splitting				
Detecting significant aortic stenosis[19]	50	79	NS	NS
Loud P_2				
Detecting pulmonary hypertension in patients with mitral stenosis[20,21]	58-96	19-46	NS	NS
Detecting pulmonary hypertension in patients with cirrhosis[22]	38	98	**17.6**	NS
Palpable P_2				
Detecting pulmonary hypertension[20]	96	73	**3.6**	**0.05**
Absent or Diminished S_2				
Detecting significant aortic stenosis in patients with aortic flow murmurs[19,23-26]	44-90	63-98	**3.8**	0.4

*Diagnostic standard: for *atrioventricular dissociation,* ventricles were paced independently of atria; for *atrial septal defect*, right heart catheterization; for *severe aortic stenosis*, aortic valve area <0.75 cm^2,[23] <0.8 cm^2,[25] peak gradient >50 mm Hg,[19,25] or peak velocity of aortic flow >3.6 m/s[24] or ≥4 m/s;[26] for *pulmonary hypertension*, mean pulmonary arterial pressure ≥50 mm Hg[20,21] or ≥25 mm Hg.[22]

†Definition of findings: *Definition of findings*: for *loud P_2*, splitting heard with loud second component[20] or S_2 louder at left second interspace than right second interspace;[21] the figures for fixed splitting of S_2 apply only to patients having audible expiratory splitting.

‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

NS, not significant.

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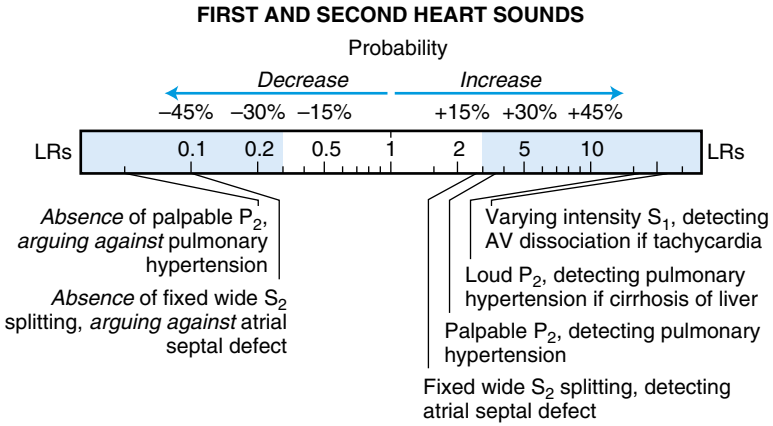

Presumably, the finding is also as accurate in patients with native rhythms. In patients with complete heart block, S_1 intensity is predictable, varying inversely with the PR interval for intervals less than 0.2 second, becoming inaudible for intervals 0.2 to 0.5 second, and becoming louder again with intervals more than 0.5 second (because the mitral valve reopens).[10]

D. PROMINENT SPLITTING OF S_1

Any delay in the closure of the tricuspid valve, the second component of S_1, accentuates splitting of S_1. This finding therefore occurs in patients with right bundle branch block (RBBB) or in LV ectopic or paced beats, all of which delay the onset of right ventricular (RV) systole and also cause wide physiologic splitting of S_2 (see later).[5,27]

How to distinguish the split S_1 from other double sounds occurring around S_1, such as S_4 + S_1 and S_1 + ejection sound, is discussed in Chapter 41.

THE SECOND HEART SOUND (S_2)

I. INTRODUCTION

The most important diagnostic feature of S_2 is its "splitting," which refers to how the aortic and pulmonic components of S_2 vary in timing during the respiratory cycle. The intensity of S_2 has less diagnostic importance. (This contrasts with S_1, in which intensity is more important than splitting.) Splitting of S_2 was first recognized by Potain in 1865, and its importance to cardiac auscultation was described by Leatham in the 1950s, who called S_2 the "key to auscultation of the heart."[28,29] The correct explanation for normal splitting—increased "hangout" in the pulmonary circulation—was discovered in the 1970s.[30,31]

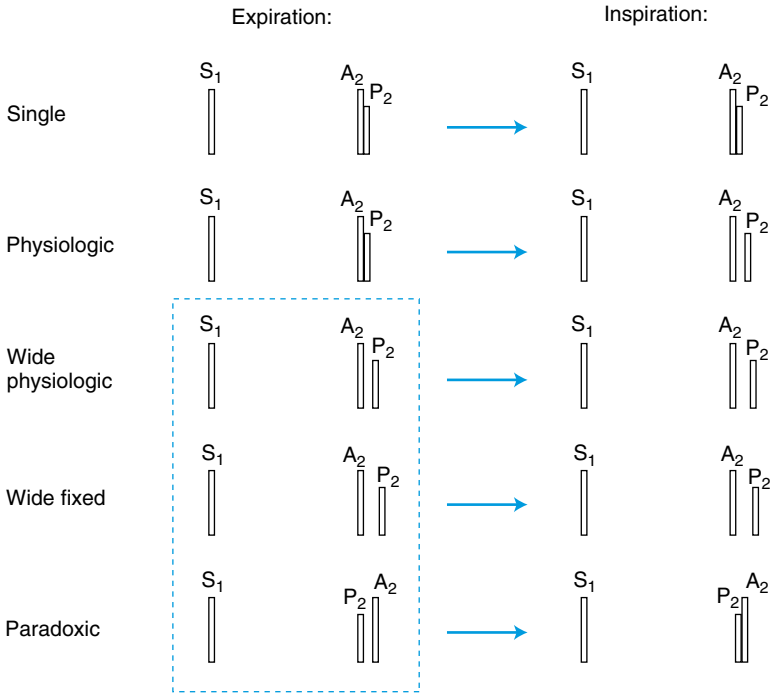

FIG. 40.1 S_2 **SPLITTING.** Splitting refers to the separation of the aortic component (A_2) and the pulmonic component (P_2) during expiration (*left column*) and inspiration (*right column*). There are two normal patterns (single and physiologic) and three abnormal patterns (wide physiologic, wide fixed, and paradoxic). The dotted lines indicate that all three abnormal forms of splitting are distinguished by having audible expiratory splitting (see the text).

II. NORMAL SPLITTING OF S_2

A. THE FINDING

In normal persons the first component of S_2 is caused by closure of the aortic valve (A_2); the second, by closure of the pulmonic valve (P_2). During inspiration the interval separating A_2 and P_2 increases by about 20 to 30 milliseconds (ms) (Fig. 40.1).[18,29,31]

Although the phonocardiogram almost always records both components of S_2, the human ear perceives them as a single sound *during expiration* in more than 90% of normal persons.[32] In normal persons *during inspiration*, the human ear either perceives two components (physiologic splitting, heard in 65% to 75% of normal adults; see Fig. 40.1)[†] or still perceives a single component (single S_2, heard in 25% to 35% of normal adults). The older the person, the more likely S_2 will be single instead of physiologic.[32,35]

[†]These two components are very close together, bordering the threshold of being perceived as a single sound. Harvey suggests mimicking the normal expiratory sound by striking a single knuckle against a tabletop and mimicking inspiratory physiologic splitting by striking two knuckles almost simultaneously.[33] Constant suggests mimicking inspiratory splitting by rolling the tongue as in a Spanish *dr* or *tr*, or saying *pa-da* as quickly and sharply as possible.[34]

In a minority of normal persons, expiratory splitting is heard in the supine position, although S_2 becomes single during expiration in these patients when they sit up.[36]

B. LOCATION OF SOUND

S_2 splitting is usually heard only in the second or third intercostal space, next to the left sternum.[35] It is sometimes heard at a slightly lower location, especially in patients with chronic pulmonary disease, and at a slightly higher location in those who are obese.[35] Splitting is not normally heard at other locations because P_2 is too faint.

C. TECHNIQUE

It is important that the patient breathe regularly in and out when evaluating S_2 splitting because held inspiration or held expiration tends to make the two components drift apart, thus making it impossible to interpret the sound.[18]

D. PHYSIOLOGY OF SPLITTING

The normal delay in P_2 results from a long "hangout" interval in the normal pulmonary circulation. (It is not because RV systole ends later than LV systole; they actually end at the same moment; Fig. 40.2.) *Hangout* means that the pulmonary circulation offers so little resistance to blood flow that flow continues for a short period even after completion of RV mechanical systole.[30,31] At the aortic valve, there is little hangout, causing flow to cease and the valve to close immediately after completion of LV contraction.

A_2 and P_2 move apart during inspiration, primarily because inspiration delays P_2 even more. Approximately half of the inspiratory augmentation of the A_2-P_2 interval is due to a further increase in the hangout interval in the pulmonary circulation. Approximately 25% of inspiratory augmentation is due to lengthening of RV systole (from increased filling of the right side of the heart during inspiration), and the remaining 25% is due to shortening of LV systole (from a reduction of filling of the left side of the heart during inspiration).[31]

III. ABNORMAL SPLITTING OF S_2

A. THE FINDING

There are three abnormalities of S_2 splitting (see Fig. 40.1):

1. WIDE PHYSIOLOGIC SPLITTING

Wide physiologic splitting means that splitting occurs during inspiration and expiration, though the A_2-P_2 interval widens further during inspiration.

2. WIDE FIXED SPLITTING

Wide fixed splitting means that splitting occurs during inspiration and expiration, but the A_2-P_2 interval remains constant.

3. PARADOXIC SPLITTING (REVERSED SPLITTING)

Paradoxic splitting means that audible expiratory splitting narrows or melds into a single sound during inspiration. Paradoxic splitting occurs because the order of the S_2 components has reversed: A_2 now follows P_2, and as P_2 is delayed during inspiration, the sounds move together.

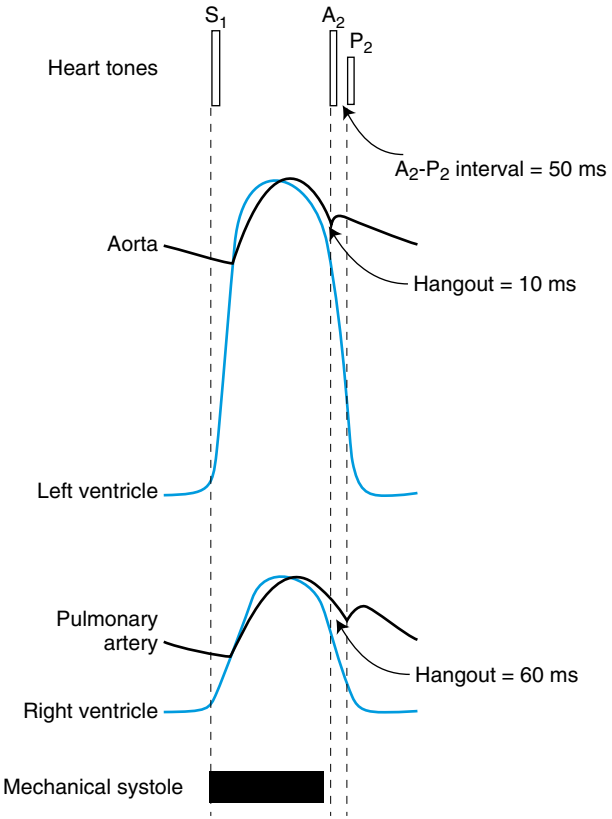

FIG. 40.2 MECHANISM OF S_2 SPLITTING. The timing of heart tones (*top*) is correlated with pressure tracings from the left side of the heart (i.e., aorta and left ventricle, *top pressure tracings*) and right side of the heart (i.e., pulmonary artery and right ventricle, *bottom pressure tracings*). The solid rectangle at the bottom of the figure depicts the duration of mechanical systole, which is the same for the right and left ventricles. A_2 coincides with the incisura (i.e., notch) on the aorta tracing, P_2 coincides with the incisura on the pulmonary artery tracing, and both sounds occur a short interval after completion of mechanical systole (the interval between the end of mechanical systole and valve closure is called hangout). On the left side of the heart, hangout is very short (10 ms, i.e., the aortic valve closes almost immediately after completion of mechanical systole). However, on the right side of the heart, hangout is longer (60 ms) because the compliant pulmonary circulation offers so little resistance to continued forward flow. The difference between these numbers explains why P_2 normally occurs after A_2 (i.e., A_2-P_2 interval in this patient = 60 − 10 = 50 ms). Changes in hangout also explain in part why splitting normally increases during inspiration and why most patients with pulmonary hypertension have a single S_2 (see the text).

B. SCREENING FOR ABNORMAL SPLITTING OF S_2

Fig. 40.1 reveals that all three abnormal second heart sounds—wide physiologic, fixed, and paradoxic—have audible splitting *during expiration* (dotted lines in Fig. 40.1). Therefore the best screening tool for the abnormal S_2 is audible expiratory splitting that persists when the patient sits up.[36-39]

TABLE 40.1 Abnormal S_2 Splitting

Splitting and Pathogenesis	Etiology
WIDE PHYSIOLOGIC	
P_2 Late	
Electrical delay of RV systole	RBBB LV paced or ectopic beats
Prolongation of RV systole	Pulmonic stenosis Acute cor pulmonale
Increased hangout interval	Dilation of pulmonary artery
A_2 Early	
Shortening of LV systole	Mitral regurgitation
WIDE AND FIXED	
Increased hangout interval or prolongation of RV systole	Atrial septal defect
Prolongation of RV systole	Right ventricular failure
PARADOXIC	
A_2 Late	
Electrical delay of LV systole	LBBB RV paced or ectopic beats
Prolongation of LV systole	Aortic stenosis Ischemic heart disease

LBBB, Left bundle branch block; *LV*, left ventricular; *RBBB*, right bundle branch block; *RV*, right ventricular. RV systole and LV systole refer to the duration of right and left ventricular contraction.

C. CLINICAL SIGNIFICANCE AND PATHOGENESIS

Table 40.1 lists the common causes of abnormal S_2 splitting.

1. WIDE PHYSIOLOGIC SPLITTING

Wide physiologic splitting may result from P_2 appearing too late or A_2 too early (Table 40.1).[18,37] The most common cause is RBBB.

In pulmonic stenosis the A_2-P_2 interval correlates well with severity of stenosis (gauged by the RV systolic pressure; $r = 0.87$, $p < 0.001$),[40] although in many patients the clinician must listen at the third interspace to hear splitting because the murmur is too loud at the second interspace.

In most patients with pulmonary hypertension the normal hangout interval disappears and S_2 is single. S_2 becomes wide in these patients only if there is associated severe RV dysfunction and prolonged RV systole.[30,31,41] Most patients with pulmonary hypertension and a wide S_2 have either long-standing severe pulmonary hypertension[30,31,41] or massive pulmonary embolism (the wide S_2 of pulmonary embolism is temporary, usually lasting hours to days).[42]

2. WIDE AND FIXED SPLITTING

Patients with atrial septal defect have wide fixed splitting of S_2, although this is true only when their pulse is regular (if the patient has atrial fibrillation or frequent extrasystoles, the degree of splitting varies directly with the preceding cycle length).[29,43] The reason S_2 is wide is not the same in every patient: in some patients, hangout is increased; in others, RV mechanical systole is prolonged.[43] S_2 is fixed

because hangout remains constant during respiration[43] and because the presence of a common left and right atrial chamber interrupts the normal respiratory variation of RV filling.[29]

In patients with audible expiratory splitting (and regular rhythm), the *absence* of fixed splitting significantly *decreases* the probability of atrial septal defect (LR = 0.1; see EBM Box 40.1), whereas the presence of fixed splitting increases the probability of atrial septal defect only modestly (LR = 2.6; see EBM Box 40.1). Patients with false-positive results (i.e., fixed splitting without atrial septal defect) commonly have the combination of RV failure and audible expiratory splitting from bundle branch block or some other cause.[18]

3. PARADOXIC SPLITTING

In elderly adults with aortic flow murmurs, the finding of paradoxic splitting does not distinguish significant aortic stenosis from less severe disease (see EBM Box 40.1).

D. S_2 SPLITTING VERSUS OTHER DOUBLE SOUNDS[39]

Other double sounds that mimic S_2 splitting include the following (see also Chapter 42):

1. S_2-OPENING SNAP

In contrast to the split S_2, the S_2-opening snap interval is slightly wider, the opening snap is loudest at the apex, and the opening snap ushers in the diastolic rumble of mitral stenosis at the apex. Patients with S_2-opening snap sometimes have a triple sound (split S_2 + opening snap) during inspiration at the upper sternal border.

2. S_2-PERICARDIAL KNOCK

In contrast to the split S_2, the S_2-knock interval is slightly wider, the pericardial knock is loudest at or near apex, and the knock is always accompanied by elevated neck veins.

3. S_2-THIRD HEART SOUND

In contrast to the split S_2, the S_2-S_3 interval is 2 to 3 times wider, and S_3 is a low-frequency sound heard best with the bell.

4. LATE SYSTOLIC CLICK-S_2

Clicks are loudest at or near apex and are often multiple. Their timing changes with maneuvers (see Chapter 46).

IV. INTENSITY OF S_2

Traditionally, a loud P_2 was regarded as a reliable sign of pulmonary hypertension, but initial attempts to confirm this teaching (mostly in patients with rheumatic heart disease) were unsuccessful. For example, in mitral stenosis patients, the loud P_2 defined either as an S_2 that is louder at the left side of the upper sternum compared with the right side[21] or as a split S_2 with a louder second component[20] did not discriminate patients with pulmonary hypertension from those without it (see EBM Box 40.1). Even when A_2 and P_2 were precisely identified by phonocardiography (e.g., A_2 corresponds to aortic incisura on simultaneous aortic pressure tracing), the relative intensities of the two components did not correlate well with pulmonary pressure.[44] Others suggested

audible splitting at the apex indicates pulmonary hypertension (because P_2 should not be heard at the apex, and any splitting at that location indicated that P_2 was abnormally loud),[32] but even this finding correlated better with the etiology of heart disease—it is common in atrial septal defect and primary pulmonary hypertension—than it did with measurements of pulmonary pressure.[41,44]

Nonetheless, one study of patients with cirrhosis did demonstrate that the loud P_2 increased probability of pulmonary hypertension (i.e., portopulmonary hypertension, LR =17.6; see EBM Box 40.1; see Chapter 8). In addition, the *palpable* S_2 in patients with mitral stenosis accurately detects pulmonary arterial pressures greater than or equal to 50 mm Hg (positive LR = 3.6, negative LR = 0.05; see EBM Box 40.1). In this study the palpable P_2 was defined as an abrupt tapping sensation coincident with S_2 at the second left intercostal space.

In patients with aortic flow murmurs, an absent or diminished S_2 increases the probability of significant aortic stenosis (LR = 3.8; see Chapter 44).

The references for this chapter can be found on www.expertconsult.com.

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